A DISSERTATION ON

TRANSESOPHAGEAL ECHOCARDIOGRAPHY STUDY IN ISCHEMIC STROKE

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CERTIFICATE

This is to certify that this dissertation entitles **"TRANSESOPHAGEAL ECHOCARDIOGRAPHY STUDY IN ISCHEMIC STROKE"** submitted by **Dr. C. ARAVIND** to The Tamilnadu Dr. M. G. R. Medical University, Chennai is in partial fulfillment of the requirement for the award of M. D. Degree Branch I (General Medicine) and is a bonafide research work carried out by him under direct supervision and guidance.

Dr.A. Ayyappan M.D.,

Professor of Medicine
Chief III Medical Unit,
Govt. Rajaji Hospital,
Madurai Medical College,
Madurai.

Dr. Nalini Ganesh. M.D.

Professor and Head
Department of Medicine,
Govt. Rajaji Hospital,
Madurai Medical College,
Madurai.

DECLARATION

I **Dr.C. ARAVIND** solemnly declare that the dissertation titled **"TRANSESOPHAGEAL ECHOCARDIOGRAPHY STUDY IN ISCHEMIC STROKE"** has been prepared by me. I also declare this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other university, board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the M. D. Degree examination in General Medicine to be held in September 2006.

Place : Madurai **Dr. C. ARAVIND**

Date :

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CONTENTS

INTRODUCTION

Information about stroke reveal that even after detailed clinical history, physical examination and bio-chemical tests, available non-invasive techniques in the evaluation, 40% of patients with ischemic stroke (80% of all stroke victims) remain without clear identifiable cause. (2002 heart and stroke statistical update, American Heart Association). This group of patients was clubbed under a sub group called cryptogenic stroke. Evaluation of these patients with semi invasive techniques like transesophageal echocardiography revealed a lot of information into the insight of the etiology of stroke with this back ground, we studied patients with stroke and the utility of transesophageal echocardiography in identifying the cause of ischemic stroke in the so called cryptogenic stroke.

AIM OF THE STUDY

Efficacy of transesophageal echocardiography in cryptogenic ischemic stroke

REVIEW OF LITERATURE

Transesophageal Echocardiography (TEE) :

Definition :

Is a diagnostic test using an ultrasound device that is passed into the esophagus of the patient to create a clear image of the heart muscle and other parts of the heart. A tube with a device called a transducer is passed down into the patient's throat and into the esophagus. The transducer directs ultrasound waves into the heart and the reflected round waves picked up by the transducer into an image of the heart.

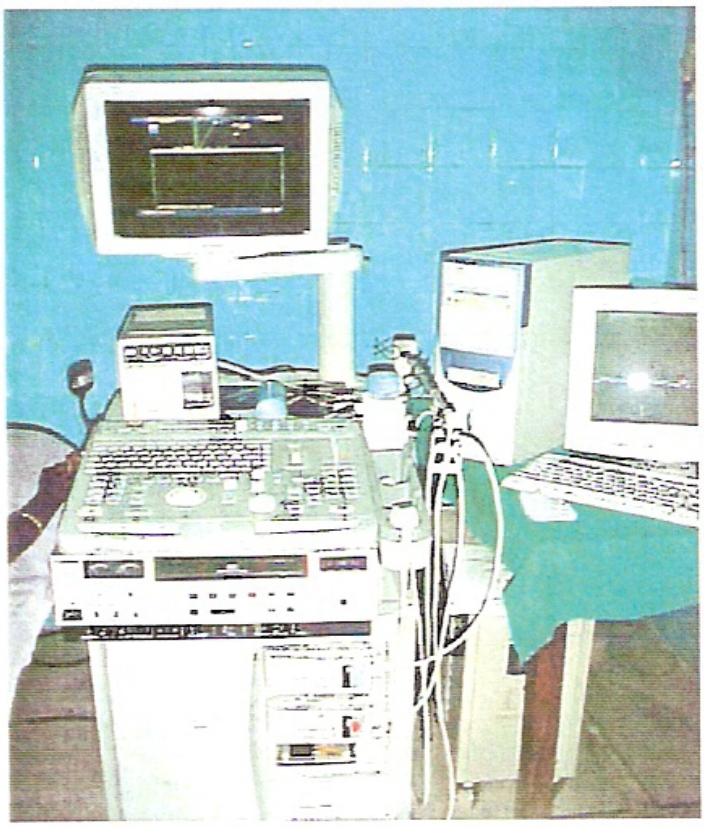
Technology :

First successful demonstration of the use of esophageal window for echocardiogram was reported in 1976 by frazin.

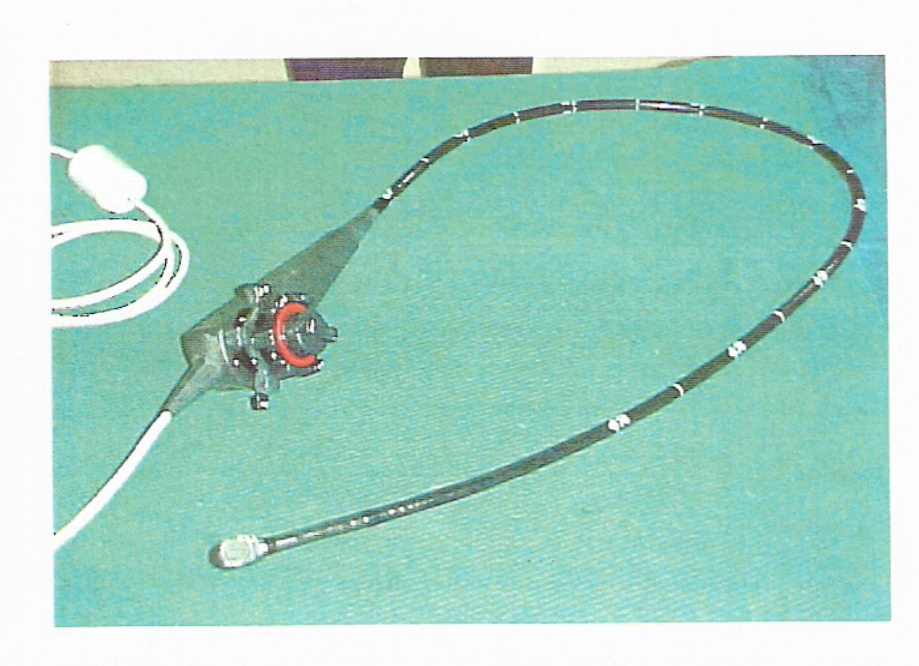
Single plan transesophageal imaging was introduced in 1990 by Dimagno et al.

Double plan transesophageal Imaging in 1982 by Souquet et al.

TRANSESOPHAGEAL ECHO CARDIOGRAPHY



TRANSESOPHAGEAL ECHO CARDIOGRAPHY PROBE

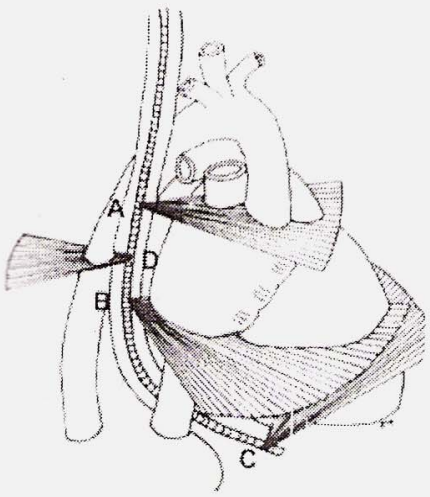


Multiplan transesophageal Imaging in 1985 by Harui and souquet.

Multiplan system provides on infinite number of planes over a full 180 rotation of a single phased array transducer.

Once intubated, the images are acquired at three levels. These are shown in figures. These include transgastric views, Lower, and Mid esophageal views and upper Esohageal views. Some operators prefer to go into the stomach straight to obtain the transgastric views first where as some prefer to go from upper esophageal to lower levels. At the end of acquiring images at these three levels, the transducer is rotated posteriorly to view the descending thoracic Aorta. Three main controls are available in the TEE probe. The probe can be rotated clockwise or counter clockwise to obtain either the views from right side or left heart. The rotatory controls have separate knobs for medial and lateral movement, anteflexion and retroflexion, and finally the array in Multiplane TEE can be moved through 180 degrees by motorized or manual controls.

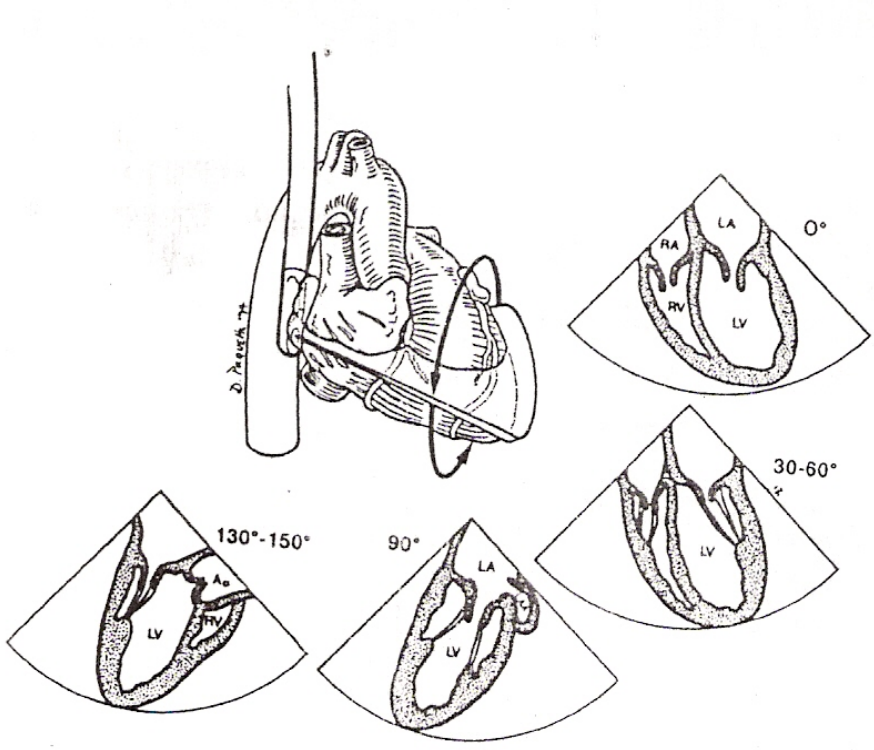
STANDARD TEE VIEWING PLANES AND VIEWS



A. Upper Esophageal views 25-30 cm
B. Mid and Lower Esophageal views 30-35 cm
   Four chamber
   Mitral commissural
   Two chamber
   Long Axis
   Ascending Aorta LAX and SAX
   Aortic valve SAX
   RV inflow – outflow
   Bicaval
C. Trans Gastric Views 35-40 cm
   SAX Mitral and PM Level
   Two chamber view
   Long axis view
   RV inflow view
   Deep Trans gastric : Apical 5 C
D. Aortic views

Modified from Otto : The Clinical Practice of Echocardiography 2nd edition 2002

LOWER AND MID ESOPHAGEAL LEVEL VIEWS OF LV



Zero degree plan is called as horizontal plane and four chamber views of both right and left heart are best obtained in this position. At around 35 to 45. Short axis views are obtained, 90 plane is called as longitudinal plane. At 135 long axis views are obtained. 180 plane is called as reverse horizontal plane and mirror image views of the horizontal plane are obtained.

Indications for Transesophageal echocardiography

TEE as a Diagnostic Tool

1. Stroke (Diagnostic yield > 50%)
2. Atrial fibrillation
3. Prosthetic valve dysfunction
4. Infective endocarditis
5. Aortic Dissection
6. Evaluation of Native valve
7. Cardiac Masses
8. ICU patient with hemodynamic instability
9. Congenital heart disease

TEE as a Procedural adjunct :

Adjunct to percutaneous procedures :

1. Percutaneous Balloon Mitral valvuloplasty
2. ASD and PFO closure
3. Interventional procedures for congenital heart disease

Adjunct to surgical procedures :

1. Heart valve repair
2. Congenital heart surgery
3. Ascending Aortic dissection repair
4. Intra operative assessment of LV function
5. Pericardial window procedures
6. Repair of Hypertrophic obstructive cardiomyopathy

VIEWS :

A - Upper Esophageal views 25 – 30 cm

B - Mid and lower esophageal views 30-35 cm

Four chamber

Mitral commissural

Two chamber

Long axis

Ascending Aorta LAX and SAX

Aortic valve SAX

RV inflow – outflow

Bicaval

C - Trans Gastric views 35-40 cm

SAX mitral and PM level

Two chamber view

Long axis view

RV inflow view

Deep trans gastric : Apical 5 c

D - Aortic view

Modified from Otto : The clinical practice of Echocardiography 2nd edition 2002.

The atrial septum is viewed from the longitudinal plane of 90. The foramen ovale the thinnest portion of the inter atrial septum is very well seen from this view. Transesophageal echocardiography has become the gold standard in the evaluation

of the inter atrial septum and pattern foramen ovale. The flap (septum primum) closes against the septum (septum secondum) before second year of life. In some patients the fusion is incomplete and the gap persists as Patent Foramen Ovale (PFO). This occurs in about 25% of general population. Figure shows the inter atrial septum and the two components. The PFOs may be small or large Counter clockwise rotation of the transducer and viewing posteriorly brings descending Aorta into focus at horizontal plane of 0^{o}. At 135 long axis views can be obtained.

Primary Indication of TEE :

A - Endocarditis :

1. High clinical suspicion
2. Clinical suspicion of abscess
3. Congestive cardiac failure
4. Prosthetic valve
5. Intracardiac device

B - Cardioembolic Source :

1. Atrial septal aneurysm

2. Aortic thrombus

3. Interatrial septal defect including patent foramen ovale.

4. Left atrial thrombus

C - Aortic Dissection

D - Preoperative valve assessment

E - Exclusion of atrial thrombus before mitral ballon valvuloplasty

F - Prosthetic valve dysfunction

G - Intraoperative application

Secondary Indications of TEE :

A - Endocarditis :

1. Low clinical suspicion

2. Prosthetic valve dysfunction

3. Native valve endocarditis without cardiac failure

B - Congenital Heart failure

C - Evaluation of ventricular function

D - Cardiac source of embolus – LV thrombus suspected

E - Evaluation of hypotensive patient

Advantage of TEE over TTE

1. No obstruction to ultrasound by chest wall structures or by lung tissue
2. Different imaging planes allow visualization of structures not seen from the precardium
3. Improved signal to noise ratio allow defection of poor echo reflective structures
4. Higher ultrasound frequencies, providing higher resolution and more detailed imaging.
5. Reduced target range for pulsed Doppler and higher sensitivity of colour flow imaging when studying posterior cardiac structures.

Contraindication :

Absolute :

1. Unco-operative or unwilling patient
2. Esophageal structure, tumour or diverticulam
3. Active upper GI bleed
4. Perforated viscus (suspected or known)

Complication :

Rare :

Sore throat, transient hypertension, hypotension, non sustained VT, hypoxia, blood tinged sputum.

Extremely rare :

Esophageal perforation, severe laryngospaim, vocal cord paralysis, ventricular tachyarrhythmias, cardiac failure, Anaphylaxis.

TEE related to our study :

TEE in potent foramen Ovale (PFO) and Atrial septal Aneurysm **(ASA) :**

The membrane of the fossa ovalis may be quite redundant and mobile and can be sufficiently large that is called interatrial septal aneurysm. Aneurysm of the atrial septum appears to involve the septum primum portion of the fossa ovalis exclusively. Both have implicated as a source of systemic embolus or atleast a site for paradoxical embolization during right to left shunting. Imaging of the nature and extent of these aneurysm and of PFO is

usually best performed between 45 and 90 with the probe tip rotated slightly rightward from the aortic valve.

Patent Foramen ovale is implicated as an important cause for various clinical syndromes. The prevalence of ‘probe’ patent foramen ovale (ie. Size 0.2 cm -0.6 cm), as evident by autopsy series is 26% (range 17% - 35%) and the prevalence of ‘pencil’ patent foramen ovale (ie size 0.6cm – 1.0 cm) is 6%. The foramen ovale has a pivotal role during embryonic fetal life. PFO is an embryological remnant of fetal circulation. It results from incomplete fusion of the flap valve of the foramen ovale with the septum secondum, a process that is usually complete by 2 years of age. But after birth it remains as a cause for paradoxical embolism, stroke, TIA, migraine, platypnea orthodexia syndrome and decompression illness in divers.

The prevalence of PFO is 46% inpatients below 55 years of age with cryptogenic stroke. The prevalence of PFO in young individuals with cryptogenic stroke is much higher than in the

elderly (21 % prevalence). According to PICS study cryptogenic stroke contributes 42% of all ischemic strokes. TEE was able to identify PFO in 39% of patients with cryptogenic stroke.

Decompression Sickness (DCS) results from formation of bubbles in body fluids as the ambient pressure is reduced. The first cause of arterial Gas Embolism (AGE) was described in a scuba diver who had an ASD. Later TEE studies showed an association between PFO and DCS in divers. Same way high altitude aviators and astronauts are prone to develop DCS. There is an increased prevalence of brain lesions in divers in the absence of recognized decompressive illness. Transcranial Doppler ultrasonography can detect right to left shunt in almost all divers with multiple brain lesions. This is most commonly due to PFO. Diving in the presence of PFO results in multiple brain lesions. Some of the diving schools recommend screening for PFO for professional divers or avid amateurs.

There is 2-5 fold increase in prevalence of migraine in PFO carriers. This may be due to small brain embolisms or due to serotonin that has bypassed the lung metabolism because of the PFO. Patients with PFO are at the risk of stroke if they undergo any major surgery. So, high risk patients need to be screened for PFO before major surgical procedure to avoid potential paradoxical embolism and the resultant major cerebro vascular events. Finally, Platypnea – orthodeoxia is a rare syndrome seen in the elderly. The affected individual becomes cyanotic and dysponeic while sitting up and this disappears when the patient lye down. A right to left shunt can be demonstrated even in PFO. The Eustachian valve may become prominent with aging. This valve directs IVC blood towards PFO. Associated pulmonary hypertension can lead to continuous arterial desaturation. PFO is commonly associated with Atrial Septal Aneursym ASA and Chiari Network which is due to incomplete resorption of septum spurium and the right valve of the sinus venosus. The prevalence of ASA is 1% and of Chairi network is 2% - 3%.as evident by autopsy studies. 83% of patients with Chairi network have PFO.

Demonstration of PFO and ASA :

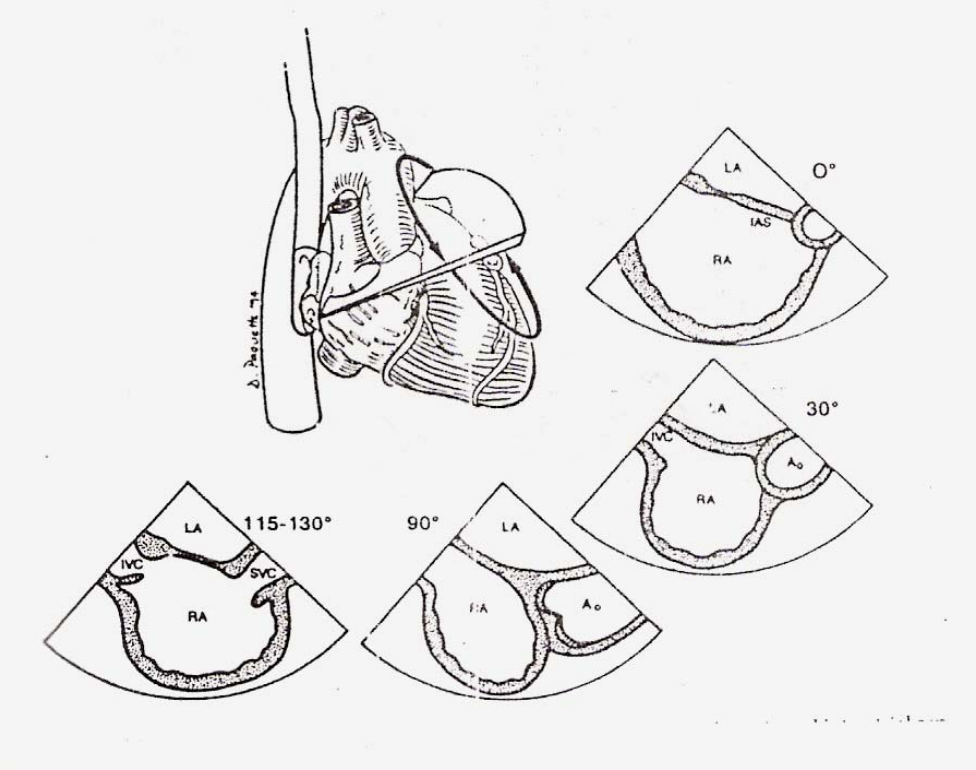
Trans throracic echocardiography, Trans esophageal echocardiography TEE, Native Tissue Harmonics (NTH) and Transcranial Doppler (TCD) are the commonly used test to identify right to left shunt. TCD cannot identify the level of this shunt. PFO can be demonstrated by TTE in 8% by NTH in 19% and by TTE in 19% of general population. Transesophagel echocardiography is the method of choice in demonstrating PFO.

This test is best done by injecting aerated colloid / saline solution through a systemic peripheral vein, preferably in the femoral vein at the end of strain phase of valsalva maneuver. When the straining is discontinued, there is sudden gush of blood that enters the right atrium and distends the right atrium. As because the left atrial chamber is still smaller for the initial 4-6 cardiac cycles, the PFO is stretched and opened. This results in transient right to left shunt at the atrial level. This causes the contrast bubbles, which entere the RA to pass through the PFO into LA. This test has excellent sensitivity.

Criteria for PFO detection includes finding of contrast in LA within three cardiac cycles after the RA opacification and the shunt is said to be significant if > 20 bubbles could be demonstrated in LA. False negative TEE may result from inadequate visualization with in the esophagus, elevated LA pressure, IVC directed flow along the IAS preventing impingment of antecubital bubbles against the IAS or an improperly done valsalva maneuver. The shunt across the PFO can be quantified as large if > 20 bubbles appear in LA and as small shunt if < 20 bubbles appear in LA. PFO can be demonstrated in 39% of patients with cryptogenic stroke and if Valsalva manure is also adopted PFO can be demonstrated in 50% of patients.

Demonstration of ASA is also important, as its presence with PFO is an important risk factor for cryptogenic stroke. 15% of patients with embolic stroke have ASA and 70% of these patients have associated PFO. But ASA is present in only 4% of the general population. ASA is defined echocardiographically as base width > 14 mm with septal excursion > 10mm.

VIEWING OF INTER ATRIAL SEPTUM

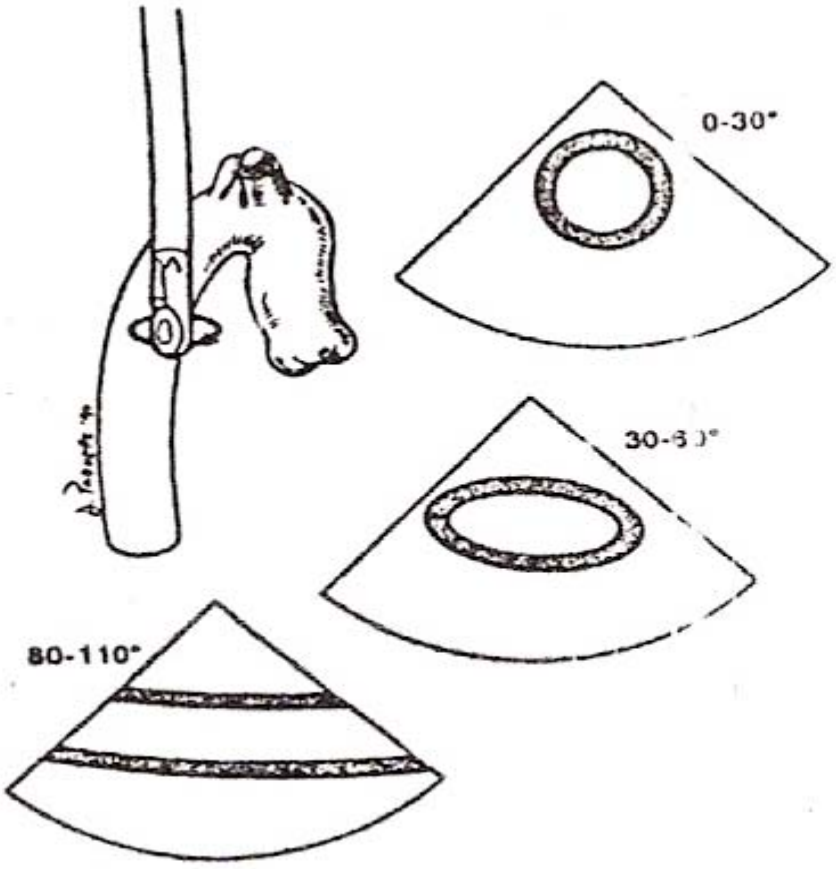


PFO needs to be closed as it increases the risk of recurrent stroke by 5 fold. The first percutaneous closure of PFO was done in 1989. Various devices are available currently and all of them are equally good. The procedural success rate is 90% - 95%. The annual recurrence rate of stroke / TIA is 4%. Patient needs antiplatelet therapy in the form of clopidogrel 75mg/day for one month and aspirin 325 mg/day for six months.

TEE in Aortic Disease :

Atheromatous disease of the thoracic aorta has been sown to be a risk factor for systemic embolization. Advent of TEE has permitted visualization of the thoracic aorta with details previously unavailable by TTE, including assessment of the intimal surface. Consequently the extent and morphology characteristic of atherosclerotic involvement of the thoracic aorta can be carefully studied particularly with Multiplan TEE karalis et al used TEE to defect atherosclerotic disease of the aorta and classified it as either

VIEWING OF AORTA



1. Simple plaque, characterized by extension from the aorta wall into the lumen less than 5 mm and maintenance of a smooth intimal surface.
2. Complex plaque is identified by extension more than 5 mm from the aortic wall into the lumen, with disruption of intimal surface.

Patient with complex atherosclerotic plague were found to have 33% incidence of an embolic event in the month preceeding evaluation. This is compared with an incidence of only 4% in group of control patient without aortic atherosclerosis evaluated by TEE.

Tunick et al reported a case control study involving 122 patients referred for TEE evaluation of unexplained stroke and TIA and 122 control patients. The presence of protruding atheromas defined as lesion extending more than 5 mm into aortic lumen was found to strongly related to the occurrence of embolic symptoms.

So many authors now recommend that complete TEE study for an unexplained source of embolism.

ISCHEMIC STROKE

Stroke :

Defined as rapid onset of focal neurological deficit, resulting from diseases of the cerebral vasculature and its contents.

In India, community surveys have shown a crude prevalence rate for stroke in the range of 200 per 1,00,000 persons, nearly 1.5% of all urban hospital admissions, 4% of all medical and around 20% of neurological cases.

Pathophysiology :

Except for lack of external elastic lamina in intracranial arteries the morphological structure of cerebral vessels in similar to other vascular bed. The arterial wall consists of advertitia, media and intima. The intima is a smooth monolayer of endothelial cells providing a non thrombotic surface to blood flow.

A cascade of complex biochemical events occurs seconds to mts after cerebral ischemia. Ischemia causes impairment of brain energy metabolism, loss of aerobic glycolysis, intracellular accumulation of Sodium and calcium irons, elease of excitotoxic neurotransmitter elevation of lactatic level with local acidosis, free

radical production, cell swelling and cell death. Many neurons undergo apoptosis after focal brain ischemia.

Complete interruption of flow causes suppression of electrical activity with 12 to 15 seconds, inhibition of synaptic excitability of control neurons after 4 mts and inhibition of electrical excitability after 4-6 mts.

Normal cerebral blood flow at rest in the normal adult brain is around 55 ml / 100 gm / mts.

First level occurs when blood flow decreases to 18ml / 100 gm / mt, brain reaches a threshold for electrical failure. At this level neurons have the potential for recovery. Second level known as threshold of membrane failure, occurs when flow decreases to 8 ml / 100 g / mt, cell death result.

Classification of Ischemic Infarction :

Pathological Classification :

1. Bland Infarction
2. Hemorrhagic Infarction

Bland Infarction :

When the cause is thrombus usually occlusion persists, preventing reperfusion of the region and resulting in pale, aremic or bland infarction.

Hemorrhagic Infarction :

Occurs when varying amount of RBC's found among the necrotic tissue. The more intense the reperfusion and the more severly damaged the capillary walls, the more confluent the hemorrhagic infarction.

Classification of Stroke :

1. Infarct - 70%
2. Hemorrhage – 27%
3. Others - 3%

70% Infarct :

1. Undetermined cause - 28%
2. Cardiac source - 14%
3. Lacune - 19%
4. Tandem arterial pathology - 4 %
5. Large artery stenosis occlusion - 5%

CARDIOEMBOLIC STROKE

Cerebrovascular events are a serious complication of a diverse group of cardiac disorder. Cardioembolic stroke are associated with substantial morbidity and mortality. Embolism of cardiac origin is 15-20 % of all ischemic stroke.

Cardiac emboli composed of platelet, fibrin, platelet fibrin, calcium microorganisms or neoplastic fragments.

Cardiac source for thrombo embolism accounts for 20% of the embolic strokes. Before the advent of Transesophageal Echo (TEE) various imaging modalities used for detection of cardiac source gave disappointed results. TEE offers superior resolution of the left and right atrium and their appendages, inter-atrial septum, aorta, patent foramen ovale, atrial septal aneurysm, vegetations, and spontaneous echo contrast. Thus TEE provides increased sensitivity for detection of various cardiac abnormalities associated with cardiac source of emboli. Thromi and vegetative lesions associated with prosthetic or native cardiac valves are also visualized with a better resolution in TEE.

Etiology of cardiac source of thrombo embolism

Definite :

- Left atrial thrombus
- Left ventricular thrombus
- Tumor
- Vegetation
- Aortic debris

Probable

- Patent foramen ovale
- Spontaneous echo contrast
- Atrial septal aneurysm

Possible

- Valvular heart disease
- Wall motion abnormality
- Mitral valve prolapse

TEE is useful in evaluation of cardiac source of embolism. TEE identifies a potential cardiac source of embolism in approximately 60% of the overall population compared with only 15% by Transthoracic Echocardiography TTE. Even in patients without

clinical heart disease, TEE identifies abnormalities up to 39% of patients with cerebral ischemic events. Pearson et al identified a potential source for embolism in 57% of the study population with TEE when compared with the TTE, which identified only 15%. Both the techniques had a similar rate of identifying apical thrombus and mitral valve prolapse. Overall TEE identified abnormalities in 39% of patients with no cardiac disease versus 19% for transthoracic echocardiography.

Left atrial thrombus :

The obvious advantage of TEE is the proximity of the left atrium to the transducer location in the esophagus. In transthoracic Echocardiography the left atrium is frequently in the far field, in which attenuation of the ultrasound signal hampers the identification of thrombus. Decisions regarding anticoagulation of patients, timing of cardioversion and assessment for percutaneous balloon mitral valvuloplasty are now more comprehensibly assessed with TEE. The left atrial appendage is visualized at the basal level using anteflexion at o degree.

Gender Raugh et al found that TEE was superior to TTE for detection of a cardiac source of embolism in patients with acute ischemic stroke, 41% versus 5% factors associated with greater likelihood of such cardiac sources for embolism included left atrial enlargement, atrial fibrillation and a younger age.

The sensitivity for detection of left atrial thrombi is 100 % specificity is 99% with the positive predictive value is 86% and negative predictive value is 100%. TEE is accurate in detecting and excluding the left atrial thrombi.

Left Ventricular thrombus :

TEE is useful in detection of LV thrombus. In mid esophageal view LV is shortened. This is overcome by the use of multiplane TEE. The sensitivity and specificity of this technique is 80 to 90%. By rotation of the transducer towards the LV help in identifying the apex. Narrowing the sector using zoom option or switching to frequency mode are also helpful. Transgastric view will also be helpful in visualizing the apex using the probe unflexed. If the transducer is rotated to 90, the apex has been in a

longitudinal orientation. A deep trans gastric view is also used where the orientation is similar to the trans thoracic view. TEE is much helpful in identification, localization, and assessment of size, calcification, mobility and attachement to the ventricular wall.

Tumor :

TEE is useful in identifying of atrial tumors like Myxoma and differentiation of Myxoma from atrial thromus. Papilloma, Fibroma and Carcinoma are the most common source of emboli. Tumours may enter atria via the inferior vena cava (hepatocellular cancer), via pulmonary veins (metastatic osteosarcoma, primary pulmonary malignancies). The value of TEE is apparent in identification of these venous spreads, which cannot be identified by transthoracic echo.

Infective Endocarditis :

Infective Endocarditis vegetations are identified with high accuracy by TEE. The diagnostic yield of two-dimensional echocardiography in identification of vegetations in patients with

documented endocarditis is approximately 60%, whereas the sensitivity of TEE in this regard is 90%. TEE is useful in the demonstration of abscess formation in Infective Endocarditis. The size, mobility and the rate of progress as assessed by TEE are important determinants of embolic potential.

Aortic debris :

Protruding and mobile debris appear to have the highest embolic potential. Large protrusive plaques have been identified both in the aortic arch and in the descending aorta in patients with unexplained embolic phenomenon by TEE.

PFO and ASD :

Patent foramen ovale and atrial septal defects are known to occur in a significantly higher percentage of patients with cerebral ischemia. Paradoxical emboli are responsible for the events. TEE is superior when compared to TTE in evaluation of ASD. With the injection of 10ml of agitated saline into an arm vein, the sensitivity and specificity of this technique is 100%. Maneuvers to

increase the right atrial pressure such as cough or valsalva may be necessary to demonstrate shunting at patent foramen ovale. It is also found in the majority of patients with atrial septal aneurysm and cerebral ischemia. PFO is also independently associated with non-lacunar stroke especially in young patients.

Spontaneous echo contrast :

Spontaneous echo contrast is a demonstration of smoke like swirling reflectance of intracardiac blood flow, most commonly seen in low flow states. Most common clinical setting in which spontaneous echo contrast found is atrial fibrillation and mitral stenosis. Spontaneous echo contrast is also seen in the left ventricle in patients with severe left ventricular dysfunction as well as in the right side of the heart. The finding of spontaneous echo contrast, particularly in the left atrium has been shown to occur in a marker for blood stasis and subsequent thrombus formation. This finding in patients with nonvalvular atrial fibrillation identifies a group of patients at high risk for subsequent embolic events.

Atrial septal aneurysm :

An atrial septal aneurysm is defined as a redundant interatrial septum usually located in the region of the fossa ovalis or sometimes involving the entire interatrial septum, which bulges 15 mm or more beyond the plane of the atrial septum at some time during the cardiac cycle into either the right or the left atrium. This abnormality is reportedly found in only a small percentage of all transthoracic echocardiograms. Case reports have identified patients particularly younger individuals, with cerebral ischemic events and no abnormalities other than atrial septal aneurysms. In TEE, atrial septal aneurysm are identified in approximately 4% of all patients studied. In patients with unexplained cerebral ischemia, however, it may be identified in up to 15% of this population. These abnormalities provide a nidus for thrombus formation and blood stasis and therefore constitute a risk factor for cerebral embolization. In patients with unexplained cerebral ischemia, atrial septal aneurysms are associated with atrial septal defects or patent foramen ovale in up to 75% of the cases. There

is high rate of recurrent stroke or transient ischemic attack in patients with atrial septal aneurysm with or without shunt.

Most common cause in older individual is atrial fibrillation, accounting two thirds of emboli of cardiac origin. Other cardiac conditions with high embolic potential include acute myocardial infarction, coronary artery disease with LV dysfunction, Rheumatic MS, Infective endocarditis, DCM, Cardiac tumours, mechanical prosthetic heart valves. Low embolic risk disorders include PFO, ASA, Aortic thrombus, hypertropic cardiomyopathy, LV aneurysm, Mitral valve prolapse, Aortic valve calcification, calcific Aortic stenosis.

Let us consider about cause related to our study ie. Coronary artery disease with LV dysfunction Rheumatic MS with or without Atrial fibrillation, ASA, PFO, Aortic thrombus, hypertropic cardiomyopathy.

Coronary Artery Disease :

Almost all episodes of embolism occur within 3 months following Acute myocardial infarction, with 85% of emboli following first month. A decreased ejection fraction is an independent predicator of an increased risk of stroke following myocardial infarction.

Rheumatic Mitral Stenosis :

Most cases of mitral stenosis are caused by Rheumatic heart disease. Systemic embolic occurs in 10-15% of patients with mitral stenosis, with 60-75 % having cardioembolic cerebral ischemia.

Atrial fibrillation :

Incidence of thrombo embolism in patient with AF is 4-8 %. Patients with nonvascular AF, leading source of cardioembolic infarction in older adults have a 6 fold increase in stroke incidence. Patient with Rheumatic AF have 17 fold increase in stroke incidence.

Patent Foramen Ovale – PFO

Before birth, a channel exists between the two upper chambers of the heart. This channel is called foramen ovale. This allows oxygenated blood received from the mother to flow from the venous circulation to the arterial circulation. Following birth, the lungs unfold and pressures in the circulatory system change, usually causing the channel to close. However, in about 25% of people, this channel does not close. In this case, the foramen ovale is said to remain patent. People with this open channel are said to have a PFO.

Significance of finding a PFO in a patient with stroke remain unclear, although several studies have supported the notion that a high incidence of PFO exist in patient with cryptogenic stroke. Incidence of PFO is autopsy series has ranged from 13 to 35 %. In a study comparing 60 adults with ischemic stroke under age 55 to 100 normal control, PFO was prevalent in 40% of stroke patient versus only 10% of control. Prevalence of PFO was even higher among those with no other identifiable cause of stroke.

Atrial septal Aneurysm - ASA :

Out pouching of greater than 10-15 mm of the interatrial septum into either the left or right atrium. Clinical relevance of ASA remain unclear, although it may possibly be associated with a high risk of cardioembolic stroke. An association between ASA and stroke was found in another study of 23 consecutive adult patient with ASA. 52% of the patient had suffered a throboemobolic cerebrovascular event.

Possible cause of stroke in ASA include embolism may occur secondary to paradoxical embolism of venous throbi, migration of a thrombus formed on the left side of the ASA or right sided thrombus farming on the ASA and embolizing through right to left shunt.

Cryptogenic Infarction or Infarct of Undetermined cause :

Despite the large number of established source of emboli, the point of origin cannot be determined. 30% of Ischemic infarct group come under this category.

There are three main reasons for this,

1. No appropriate laboratory studies are performed.
2. Improper timing of the appropriate lab studies
3. Normal findings are reached despite appropriate lab studies performed at appropriate time. 40% of the cause of ischemic stroke of undetermined cause fall in this category.

Emerging technologies have led to the suggestion that some of the cyptogenic infarct cause may be explained by hematological disorder like hypercoaguable state. Protein C, Free protein S, lupus anticoagulant or anticardiolipin antibody abnormalities. Other have implicated paradoxical emboli through PFO or emboli from aorta and from ASA.

If extensive evaluation fails to disclose the origin, the odds still favour a source in left heart, not infrequently the diagnosis of cerebral is made at autopsy without finding a source. Nevertheless, in some cases, even when studied carefully postmortem, no source of embolic material can be discovered.

MATERIALS AND METHODS

Setting : Inpatient of Government Rajaji Hospital, Madurai

Design of study : Observational study

Period of study : One year from October 2004.

Sample Size and Selection of study subjects : Fifty three CT Brain proved conscious ischemic stroke patients admitted in our department are included in this study. This study included 31male and 22 female patients.

Details of Study Subjects :

All fifty three CT brain proved conscious ischemic stroke patients were underwent detailed history and physical examination. History include fever, meningeal symptoms, weight loss, trauma, transient ischemic attack, and joint swelling or pain and symptoms related to cardiac and respiratory conditions. And any exposure to sexual transmitted disease or extramarietel contact. Whether he or she is a known case of diabetes, hypertension, pulmonary tuberculosis, congenital heart disease, Rheumatic heart disease, ischemic heart disease or cancer patient.

Physical examination include pulse rate, rhythm, character, felt in all palpable peripheral arteries, whether there is an thrill or bruit and blood pressure of both upper limb and lower limb.

All the subject had routine blood investigation like total count, differential count, hemoglobin level, renal function test and blood sugar level.

A 12 lead electrocardiogram is taken and transthoracic echocardiogram is also done for all patients.

All ischemic stroke patients of undetermined cause undergone rapid test HIV 1 and 2 and lipid profile.

Then atlast all patients underwent transesophageal echocardiography evalvation with multiplane capabilities using ALOKA SSD 2000 machine.

Preparation of Patients before TEE :

Eight hours fasting is adviced. Mild sedation before the procedure and an anesthetic agent is sprayed into the back of the throat in order to suppress the gag reflex. Transesophageal echocardiographic examination generally last 15-20 mts. A special viewing tube called an endoscope, containing a tiny transducer is passed through the mouth and into the esophagus. It is carefully moved until it is positioned directly next to the heart. Essentially a modified microphone, the transducer directs ultrasound waves into the heart. Some of which get reflected back

to the transducer. Different tissue and blood all reflect ultrasound waves differently. These sound waves can be translated into a meaningful image of the heart, which is displayed on a monitor or recorded on paper or tape.

The following characteristics were studied in all the groups. The left atrial appendage size, presence of left atrial thrombus, atrial septal aneurysm and patent foramen ovale were noted for contrast study was done in all the patients by injecting agitated saline through right upper limb peripheral vein. The presence of even a single bubble in the left atrium within the first 3 to 5 cardiac cycles was taken as an evidence for the presence of right and left shunting across the patent foramen ovale. The inter atrial septum was imaged in atleast 3 views, long axis plane, short axis plane, and the bi-cameral view.

Presence of interatrial septal excursions were also interrogated to rule out atrial. Septal aneurysm. A definite atrial septal aneurysm was diagnosed when there was atleast 1.1 cm

excursion into either left atrium or right atrium with a base of atleast 1.5 cm.

All the patients underwent imaging of the ascending, arch and descending aorta for the presence of atheromas, mobile masses protruding into the lumen.

After care :

After the test, it is important to refrain from eating or drinking until the gag reflex has returned otherwise the patient may accidentally inhale some of the food or beverage.

RESULTS

Among 53 ischemic stroke patients 11 had Rheumatic mitral stenosis with and without atrial fibrillation, 7 were female and mean age in the group was 30.1 + 6.2 years and 11 were categorized as cryptogenic ischemic stroke patient (without TEE) and 31 were categorized as other causes ie. having risk factors like diabetes, hypertension, ischemic heart disease and hypertropic cardiomyopathy.

Among the Rheumatic mitral stenosis patients 4 had atrial fibrillation and 6 had left atrial thrombus

Among the other causes patients, 5 were known case of diabetes, 18 were known case of hypertension and diabetes, 2 were known case of ischemic heart disease and 1 was hypertropic cardiomyopathy.

Percentage of ischemic stroke patients with risk factors like diabetes, hypertension, Rheumatic heart disease, ischemic heart disease with LV dysfunction and hypertrophic cardiomyopathy was 79.3%.

Percentage of ischemic stroke due to Rheumatic heart disease with and without Atrial Fibrillation was 20.7%.

Percentage of ischemic stroke due to hypertension was 9.4%.

Percentage of ischemic stroke due to diabetes was 9.4 %

Percentage of ischemic stroke due to hypertension and diabetes was 34%.

Specific to our study :

Percentage of atrial septal aneurysm in our study was 7.5%

Percentage of Aortic thrombus in our study was 5.6 %

Percentage of patent foramen ovale in our study was 3.7 %

Percentage of Atrial septal aneurysm among cryptogenic stroke patient 36.4%

Percentage of Aortic thrombus among cryptogenic stroke patient was 27.3 %

Percentage of patent foramen ovalve among cryptogenic stroke patient was 18.2 %

In cryptogenic stroke patient rheumatic heart disease, diabetes, hypertension and ischemic heart disease were ruled out.

All cryptogenic stroke patient were under 50 years, mean age 38.5 years.

None of the patient was smoker or alcoholic. In all cryptogenic stroke patients lipid profile was normal and rapid test HIV was negative.

Among the previously diagnosed as 11 cryptogenic stroke patients, 9 patients the cause was identified by TEE. (4 had atrial septal aneurysm, 3 had aortic thrombus and 2 had patent foramen ovale). In 2 patients the cause was undetermined. So these patients should undergo further investigations like coagulation profile.

But transthoracic echocardiography was normal in the 11 patients.

Therefore percentage of ischemic stroke of undetermined cause without TEE – 20.7% and percentage of ischemic stroke of undetermined cause TEE was 3.7 % only.

Therefore in the so called cryptogenic ischemic stroke 9 of the 11 patients had positive finding in TEE which were not picked up by transthoracic echocardiography.

Therefore, Transesophageal echocardiography play an important role in identifying the cause in patients who were previously diagnosed as cryptogenic ischemic stroke patients.

DISCUSSION

In 1984, Hagen et al reported about 27%. Prevalence of atrial septal malformation in an autopsy series of 965 hearts believed to be normal. The prevalence of septal abnormalities declined from 34% during the first 3 decades of life to 20% in the ninth decade of life.[6]

Lynch et al performed contrast echo evaluation in 100 patients with no history of stroke and 60 patients with ischemic stroke and found a 10%. Prevalence of PFO in the control group and as high as 40% in the stroke patients.[7]

Pearson et al established the superiority of Transesophageal echocardiography over transthoracic echocardiography in 79 patients of unexplained stroke, where only 15% had potential cardiac source of embolism in transthoracic echocardiography but about 57% had some of evidence of cardiac source when examined with transesophageal esophageal echocardiography.[9] The abnormalities detected were atrial septal aneurysm with patent

foramen ovale, left atrial appendage thrombus and left atrial spontaneous echo contrast.

Pearson et al in yet another study found that atrial septal aneurysm was diagnosed more frequently in patients with stroke 15% vs 4% (normal). [9,28]

In multiple studies by Karalis et al[10], Katz et al[11], Tunick et al[12], Vaduganathan[13], there was a positive correlation of the presence of aortic atheromo with stroke.

Di Pausquale in his studied showed atrial septal aneurysm a potential cause of cardioembolic stroke. [29]

Hausmann et al , mugge et al, study compares the value of transthoracic and transesophageal contrast echo cardiography for detecting a patent foramen ovale. A total of 238 patients were studied. 74 patients with a history of otherwise unexplained ischemic stroke or transient cerebral ischemic attack (Group A); 48 with a history of similar episodes explained by other cardiac abnormalities (group B) and 116 with no embolic events (group c)[24,27]

TEE play an important role in diagnosing atrial septal aneurysm was showed in two studies.[30,31]

A PFO was detected by contrast transesophageal echocardiography in 50 of 238 patients compared with 45 patients by transthoracic. In a subgroup of 198 patients, tranesophageal echocardiography results could be compared with transthoracic echocardiography findings. Contrast transthoracic echo cardiography detected a PFO in 15 patients compared with contrast transesophageal echocardiography which demonstrated a PFO in 44 of 198 patients.

Prevalence of PFO of TEE was 22, 21 and 22% in groups A,B and C respectively. Thus, contrast transophageal echocardiography are significantly superior to transthoracic echocardiography for detected patient foramen ovale. Prevalence of PFO is significantly increased in young adults with otherwise unexplained ischemic stroke.

In our study also we found high percentage of patients with inter atrial septal abnormalities like atrial septal aneurysm and patent foramen ovals, plaques in the aorta were also present in

quite good number of patient in the so called cryptogenic ischemic stroke group.

Cabanes et al found in his study that among cryptogenic ischemic stroke patients 39% had atrial septal aneurysm which was similar in our study 36.4%. However the percentage of patent foramen ovale was much higher in their study group 56.3%. Unlike in our study 18.2%[14]

Mowla Ashkan et al in his study of 98 patients with ischemic stroke found out abnormalities in 94.8% of patients. Using transesophageal echocardiography. But in our study of 53 ischemic stroke patients we found abnormal transesophageal echocardiography in the form of valvular pathology, atrial septal aneurysm, patent foramen ovale and aortic atherumas in about 20 patients. 37.7%.[15]

Bruno censori found in his study that an early TEE doesnot seem to increase substantially the detection of atrial thrombi or spontaneous ECHO contrast in patients with a first stroke attack of cryptogenic or Lacunar nature. Therefore, this examination can be

carried out when the patient conditions are stable and without overloading the cardiovascular laboratory daily schedule.[26]

Nishide showed in his study the most common cardiac abnormalities in ischemic stroke studied by two-dimensional echocardiography.[32]

Shyu pointed out the role of TEE in the diagnostic assessment of cardiac sources of embolism in patients with acute ischemic stroke. [33]

Andreas Mugge found in his study that TEE is superior to TTE approach in the diagnosis of ASA. The most common abnormalities associated with ASA are interatrial shunts in particular patent foramen ovale. In this study, patient with ASA showed a high frequency of previous clinical events compatiable with cardiogenic embolism, in a significant subgroup of patients, ASA appears to be the only source of embolism, as judged by TEE. Our data are consistent with the view that ASA is a risk factor for cardiogenic embolism, but thrombi attached to ASA as detected by TEE are apparently rare. [25]

However the diagnostic yield of transesophageal echocardiography in patients with ischemic stroke of no known cause that is cryptogenic ischemic stroke was high as 81.8% (9 out of 11 patients) in our study.

To conclude, we recommend the routine use of transesophageal echocardiography in patients with ischemic stroke to find out the possible cardiac source of embolism with special emphasis in patients with no other possible or probable cause of ischemic stroke, cryptogenic stroke.

Limitation of the Study :

Because of the financial constraints in the institution and poor socio economic status of patients in our study population, exhaustive workup of the coagulation profile was not done.

Similarly carotid Doppler and intimal thickness measurement were not routinely done in these patients.

Eventhough the mean age of the study group in cryptogenic ischemic stroke was 38.5 years and carotid abnormalities are not routinely expected in this age group, it however is a limitation in our study.

CONCLUSION

To conclude, transesophageal echocardiography evaluation should be an integral part in diagnostic workup of patients with cryptogenic stroke. Our study showed atrial septal aneurysm, positive contrast study and aortic thrombus in 9 out of 11 number of patients.

SUMMARY

Among 53 concious ischemic stroke patients 11 had Rheumatic mitral stenosis with and without atrial fibrillation and left atrial thrombus, 11 were categorized as cryptogenic ischemic stroke patients and 31 were categorized as other causes patients.

All cryptogenic stroke patients were under 50 years, mean age was 38.5 years, None of the patient was smoker or alcoholic. In all cryptogenic patients lipid profile was normal and rapid test HIV was negative. Among the previously diagnosed as 11 cryptogenic stroke patients, 9 patients the cause was identified by TEE. In 2 patients the cause was undermined, so these patients should undergo further investigations like coagulation profile.

Therefore transesophageal echocardiography play an important role in identifying the cause in patients who were previously diagnosed as cryptogenic ischemic stroke patients.

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PROFORMA

TRANSESOPHAGEAL ECHOCARDIOGRAPHY STUDY IN CRYPTOGENIC ISCHEMIC STROKE

Name : Age : Sex :

History : Fever, Meningeal symptoms, weight loss, trauma, transient ischemic attack, joint swelling or pain symptoms related to cardiac and respiratory conditions.

Diagnosis :

CT Scan :

HT : Diabetes :

Rheumatic Heart Disease :

TIA :

Pulse rate :

Systolic BP : Diastolic BP :

Murmur :

Random blood sugar : Urea :

Creatinine :

ECG : TTE :

Inter atrial septum :

Contrast TEE study :

Left atrial appendage : Descending Aorta :

Atheroma : Left atrial clot :

MASTER CHART

S.No.	Name	Age	Sex	Diagnosis	CT Scan	HT	DM	RHP	TIA	PR	SBP	DBD
1.	Pappathy	46	F	L.Hemiplegia	RMCA	+	+	-	-	104	150	100
2.	Pandiammal	35	F	R.Hemiplegia	LMCA	-	-	+	-	106	130	100
3.	Mangayarkani	24	F	L.Hemiplegia	RMCA	+	-	-	-	80	140	90
4.	Veerammal	40	F	L.Hemiplegia	RMCA	-	-	-	-	76	130	90
5.	Karuppasamy	35	M	R.Hemiplegia	LMCA	+	+	-	-	90	140	100
6.	Poomari	36	F	R.Hemiplegia	LMCA	-	-	-	-	100	130	90
7.	Gomathiammal	65	F	R.Hemiplegia	LMCA	+	+	-	-	76	140	90
8.	Anantha jothi	33	F	R.Hemiplegia	LMCA	+	-	-	-	80	130	90
9.	Parthasarathy	65	M	R.Hemiplegia	LMCA	-	-	-	-	80	140	90
10.	Vedamoorthy	50	M	L.Hemiplegia	RMCA	-	+	-	-	82	140	90
11.	Immanuel	24	M	L.Hemiplegia	RMCA	-	-	-	-	90	130	90
12.	Rajendran	40	M	L.Hemiplegia	RMCA	+	-	-	-	90	140	90
13.	Paulraj	45	M	R.Hemiplegia	LMCA	-	-	-	-	84	150	100
14.	Chandrasekar	56	M	L.Hemiplegia	RMCA	+	+	-	-	90	130	80

15.	Marimeena	61	F	R.Hemiplegia	LMCA	+	+	-	-	90	120	80
16.	Masanam	48	M	R.Hemiplegia	LMCA	-	-	-	-	80	130	90
17.	Boominathan	70	M	L.Hemiplegia	RLMCA	+	-	-	-	90	130	90
18.	Samraj	38	M	L.Hemiplegia	RMCA	-	-	-	-	76	146	100
19.	Gurusamy	47	M	R.Hemiplegia	LMCA	+	+	-	-	86	134	90
20.	Veerathevar	57	M	R.Hemiplegia	LMCA	-	+	-	-	90	130	90
21.	Mayan	59	M	R.Hemiplegia	LMCA	-	+	-	-	92	140	90
22.	Vasanthakumari	29	F	R.Hemiplegia	LMCA	-	-	+	-	100	140	80
23.	Roshanbeevi	70	F	L.Hemiplegia	RMCA	+	+	-	-	76	130	80
24.	Meenalochani	60	F	R.Hemiplegia	LMCA	+	-	-	-	80	140	90
25.	Ammapillai	32	M	L.Hemiplegia	RMCA	-	-	+	-	86	120	90
26.	Uma rani	36	F	L.Hemiplegia	RMCA	-	-	-	-	80	110	70
27.	Amaravathy	30	F	L.Hemiplegia	RMCA	-	-	+	-	90	130	90
28.	Pandi	40	M	L.Hemiplegia	RMCA	-	+	-	-	80	120	90
29.	Sagundala	60	F	R.Hemiplegia	LMCA	+	+	-	-	86	120	80
30.	Vellammal	39	F	R.Hemiplegia	LMCA	-	-	-	-	92	130	80
31.	Alagar	40	M	L.Hemiplegia	RMCA	-	-	+	-	82	130	80

32.	Sankarammal	34	F	L.Hemiplegia	RMCA	-	-	+	-	72	140	96
33.	Gomathy	56	F	R.Hemiplegia	LMCA	+	+	-	-	76	140	90
34.	Gulam	52	M	L.Hemiplegia	RMCA	+	+	-	-	90	130	90
35.	Vedamuthu	70	M	R.Hemiplegia	LMCA	+	+	-	-	90	150	90
36.	Pathapriya	22	F	R.Hemiplegia	LMCA	-	-	+	-	76	140	80
37.	Murugesan	62	M	R.Hemiplegia	LMCA	+	+	-	-	86	130	90
38.	Ammavasai	57	M	L.Hemiplegia	RMCA	+	+	-	-	80	140	90
39.	Pappammal	54	F	L.Hemiplegia	RMCA	+	+	-	-	86	120	80
40.	Sasikumar	40	M	R.Hemiplegia	LMCA	+	+	-	-	920	130	80
41.	Muthu	40	M	L.Hemiplegia	RMCA	-	-	-	-	92	140	70
42.	Sundran	40	M	L.Hemiplegia	RMCA	-	-	-	-	76	145	76
43.	Radha	62	F	L.Hemiplegia	RMCA	-	-	-	-	86	136	80
44.	Raja	46	M	L.Hemiplegia	RMCA	-	-	-	-	86	130	90
45.	Balan	27	M	L.Hemiplegia	RMCA	+	+	-	-	80	126	80
46.	Vasu	40	M	R.Hemiplegia	LMCA	-	-	-	-	70	130	90
47.	Malliga	28	F	L.Hemiplegia	RMCA	-	-	+	-	110	100	70
48.	Jansi	33	F	R.Hemiplegia	LMCA	-	-	+	-	106	110	80

49.	Nuthan	24	M	L.Hemiplegia	RMCA	-	-	+	-	120	120	70
50.	Rajendran	30	M	R.Hemiplegia	LMCA	-	-	+	-	110	100	70
51.	Madasamy	55	M	R.Hemiplegia	LMCA	+	+	-	-	90	130	100
52.	Kannan	63	M	L.Hemiplegia	RMCA	+	+	-	-	90	126	96
53.	Arjun	40	M	L.Hemiplegia	RMCA	-	-	-	-	80	130	96

S.No.	Sugar	Urea	Crea tinine	ECG	TTE	IAS	Bubble study	LAA	Des. Aorta	Atheroma	LA Clot
1.	120	50	0.7	SR/WNL	N	I	-	2.2x1.9	2x1.9	-	-
2	75	24	1	SR/WNL	MS severe	I	-	3.9x2.2	1.9x1.2	-	-
3	79	50	1	SR/WNL	N	I	-	2.5x1.3	1.7x1.2	-	-
4	140	32	0.6	SR/WNL	N	ASA	-	2.0x1.7	1.8x1.0	-	-
5	102	25	0.9	SR/WNL	N	I	-	1.7x2.8	2.2x1.2	-	-
6	102	29	1	SR/WNL	N	I	+	3.3x1.6	1.8x1.1	-	-
7	202	30	1	SR/WNL	N	I	-	2.2x1.8	2.3x1.2	-	-
8	136	30	0.8	SR/WNL	N	I	-	2.0x1.3	1.8x1.0	-	-
9	120	30	0.9	SR/WNL	N	I	-	2.1x1.3	1.8x1.1	-	-
10	126	30	0.7	SR/WNL	N	I	-	2.2x1.2	1.3x1.1	-	-
11	120	30	0.7	SR/WNL	N	I	-	2.0x1.3	2.0x1.2	-	-
12	130	35	1	SR/WNL	N	I	-	2.3x1.3	1.7x1.2	-	-
13	106	25	0.7	SR/WNL	N	ASA	-	2.0x1.2	2.3x1.2	-	-
14	206	30	0.8	SR/WNL	N	I	-	2.4x1.3	2.1x1.2	-	-

15	140	40	1	SR/WNL	N	I	-	2.7x1.2	1.9x1.2	-	-
16	120	36	1	SR/AWMI	RWMA Moderate LV dysfunction	I	-	2.6x1.1	1.9x1.2	-	-
17	118	32	0.7	SR/WNL	N	I	-	2.2x1.9	2.1x1.2	-	-
18	127	35	0.8	SR/WNL	N	I	-	2.3x1.9	2.2x1.6	-	-
19	212	30	1	SR/WNL	N	I	-	2.3x1.2	2.4x1.5	-	-
20	106	28	0.7	SR/WNL	N	I	-	3.1x1.1	2.6x1.7	-	-
21	200	25	1	SR/WNL	N	I	-	2.6x1.2	2.7x1.6	-	-
22	106	30	0.6	SR/LAE	MS Severe	I	-	2.7x1.1	3.1x1.6	-	-
23	163	32	0.7	SR/WNL	N	I	-	2.7x1.6	2.6x1.2	-	-
24	112	26	0.8	SR/WNL	N	I	-	3.1x1.6	2.7x1.6	-	-
25	160	25	1.1	SR/LAE	MS Severe MR Grade II	I	-	2.6x1.2	2.5x1.7	-	-
26	120	40	1	SR/WNL	N	I	-	2.7x1.7	2.1x2.0	-	-
27	120	30	0.7	SR/LAE	MS Severe	I	-	2.6x1.6	2.2x1.6	-	-
28	140	31	0.7	SR/WNL	N	I	-	3.1x1.6	2.1x1.2	-	-
29	155	30	0.6	SR/WNL	N	I	-	3.0x1.5	2.1x1.3	-	-
30	150	40	0.8	SR/WNL	N	ASA	-	2.9x1.6	2.2x1.6	-	-
31	120	41	1	SR/LAE	MS Severe	I	-	2.8x1.6	1.9x1.1	-	-

32	120	40	1	SR/LAE	MS Severe	I	-	2.7x1.2	2.1x1.6	-	-
33	140	30	0.8	SR/WNL	N	I	-	3.0x1.6	2.2x1.6	-	-
34	155	30	0.7	SR/WNL	N	I	-	2.6x1.6	2.3x1.4	-	-
35	160	30	1	SR/WNL	N	I	-	2.9x1.6	2.0x1.6	-	-
36	125	40	0.7	SR/LAE	MS Severe	I	-	2.6x1.3	2.0x1.2	-	-
37	162	35	0.6	SR/WNL	N	I	-	2.4x1.2	2.0x1.2	-	-
38	152	32	1	SR/WNL	N	I	-	2.2x1.6	2.2x1.6	-	-
39	168	40	0.8	SR/WNL	N	I	-	2.1x1.4	2.1x1.2	-	-
40	129	40	0.7	SR/WNL	N	I	-	2.2x1.6	2.2x1.2	-	-
41	168	30	0.8	SR/WNL	N	I	-	2.2x1.4	2.1x1.2	-	-
42	120	25	0.9	SR/AWM1	RWMA / Moderate LV dysfunction	I	-	2.3x1.4	2.1x1.1	-	-
43	118	30	1	SR/LVH	ASH/SAM LVOT GV 63 TR 26MM Hg	I	-	2.3x1.2	2.1x1.1	-	-
44	175	35	0.7	LVH	LVH	I	-	3.8x1.1	2.5x2.0	-	-
45	160	36	0.6	SR/WNL	N	I	-	3.6x1.0	2.4x1.6	-	-
46	120	25	0.7	SR/WNL	N	I	+	3.7x1.2	2.5x1.6	-	-
47	120	38	0.8	AF / CVR	MS Severe	I	-	3.8x2.2	2.6x1.4	-	-
48	160	40	1	AF / CVR	MS Severe	I	-	3.6x2.0	2.5x1.6	-	-

49	175	42	0.7	AF / CVR	MS Severe	I	-	3.6x2.2	2.2x1.2	-	-
50	170	30	0.6	AF / CVR	MS Severe	I	-	3.6x1.6	2.6x1.0	-	-
51	170	40	1	SR / LVH	N	I	-	2.5x1.2	2.6x1.6	-	-
52	160	35	1.1	SR / LVH	N	I	-	2.5x1.1	2.2x1.4	-	-
53	120	30	0.6	SR / LVH	N	I	-	2.4x1.6	2.1x1.2	-	-